Intimal Thickening after Placement of a Neuroform Stent

Its Incidence and Relation to Angiographic Follow-up Results of Aneurysm Embolization

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Summary

Little attention has been given to the intimal thickening of the parent artery associated with the use of Neuroform stent. The purposes of this study were are to analyze quantifyanalyze the incidence of the parent artery intimal thickening the incidence and pattern of luminal changes, to to see somedetermine possible predictors of the phenomenonof the intimal thickening, to to evaluate the its effectthe phenomenonrestenosis on the aneurysm treatment results.

We reviewed the initial and six-month follow-up angiographic images in 32 intracranial aneurysm patients treated with Neuroform stent and coilsin wide-necked aneurysm treatment. The initial embolization results were evaluated by the Raymond and Roy classification. The angiographic changes from immediate post-embolization to the six-month follow-up were classified as 'improved', 'unchanged' and 'worse'. The occurrencerates of parent artery intimal thickening was observed. Any perceivable change in the stented segment of the parent artery was considered as 'intimal thickening' and any change of ≥50% as 'significant thickening'. Fisher exact tests and logistic regression analysis were applied to determine the relation between the occurrence of the intimal thickening and several variables.

The incidence of the intimal thickening was 18.8% (6/32) and of significant thickening, 3.1% (1/32). The change in angiographic occlusion of the aneurysm was 'improved' in 40.6% (13/32), 'unchanged' in 37.5% (12/32), and 'worse' in 21.9% (7/32). Among the variables, patient's age (≥55) and follow-up angiographic results ('improved') correlated with the occurrence of the intimal thickening. Of notable finding was all six cases with intimal thickening of the parent artery were associated with 'improved' in their follow-up angiographic result.

Neuroform-associated intimal thickening usually occurs in younger patients and is frequently associated with improved angiographic result of the aneurysm embolization on follow-up.

Introduction

The Neuroform stent (Boston Scientific, Fremont, CA) has been successfully used in the stent-assisted endovascular treatment of either ruptured or unruptured, wide-necked aneurysms [1-4]. Its delivery system and stent design have been improved to facilitate easy delivery, to prevent the stent strut from herniating into the aneurysm sac, and to enhance the coil packing of the aneurysm [5]. One of the potential problems of using a stent in otherwise normal parent artery is in-stent stenosis. Recently an incidence of 5.8% has been reported [6]. The mechanism of this phenomenon is not well understood and may not have the same mechanism as the in-stent restenosis seen in patients with atherosclerotic lesions.

We recently experienced a case of in-stent stenosis in the internal carotid artery (ICA) in which a Neuroform stent was deployed to assist in the coil embolization of an ophthalmic segment aneurysm (figure 1A and 1B). The degree of stenosis was severe enough to compromise ipsilateral carotid flow. Interestingly, the residual contrast filling of the aneurysm, noted on the patient's immediate post-treatment angiogram, disappeared on the six-month follow-up angiogram. This finding prompted us to compare the initial post-treatment and follow-up angiographic features in patients treated with Neuroform stent-assisted coiling technique.

During the review process, we found that there was variable degree of mild stenosis of the stented parent artery, which could be explained as a reactive change in the vascular intima to the stent. We coined the finding as 'intimal thickening'. Specifically we looked at the incidence of the intimal thickening and its correlation to the change of the degree of aneurysm obliteration. The purposes of this study are to analyze the incidence of intimal thickening of the parent artery following implantation of a Neuroform stent, to determine possible predictors for the stenosis, and to evaluate the possible implications of the phenomenon in aneurysm treatment.

Table 1 **Baseline characteristics of the study patients and the aneurysms.**

Patients		
Total number		32
Sex (female)		30 (93.8%)
Age (years)	Mean	57
	Range	28-82
Any risk factor for atherosclerosis		12 (37.5%)
Hypertension		6
Diabetes		2
Hyperlipidemia		5
(Ex)smoker		3
Coronary heart disease		1
Aneurysms		
Total number		32
Unruptured		28 (87.5%)
Mean size (mm)		9.7 (4-20)
Location		
Cavernous segment of ICA		3
Clinoid segment of ICA		1
Ophthalmic segment of ICA		9
Superior hypophyseal artery origin		6
Posterior communicating artery origin		1
Anterior communicating artery origin		1
Vertebral artery, distal		1
Basilar trunk		2
Superior cerebellar artery origin		2
Basilar top		6

Material and Methods

Patients

From March 2005 to August 2006, 149 patients with 153 cerebral aneurysms were treated with endovascular coil embolization at our institution. Among these patients, 48 patients with 49 aneurysms had wide-necked aneurysm and were implanted with a Neuroform stent to assist in the coil embolization of the aneurysm. Thirty-two of these 48 patients had a six-month follow-up angiogram. Each of these patients had one target aneurysm treated with a stent (Table 1).

Procedure and follow-up imaging

The use of Neuroform stent to treat wide-necked cerebral aneurysms was approved by our institutional review board. Every procedure was performed after written informed consent was obtained from the patient and/or the person(s) legally responsible for the patient.

All study patients with unruptured aneurysms were pretreated with dual antiplatelet therapy. Aspirin (325 mg/day) and clopidogrel (Plavix; Bristol-Myers Squibb/Sanofi-Avantis Pharmaceuticals, New York, NY; 75 mg/day following a loading dose of 300 mg) were orally administered for not less than five days before stenting. An additional loading dose was given if the result of the platelet function test (VerifyNow Aspirin and $P2Y_{12}$ for Plavix; Accumetrics, San Diego, CA) was lower than the therapeutic level. In patients with ruptured aneurysm, a single dose of aspirin and a loading dose of clopidogrel were given immediately before

the stent placement through an oro-gastric tube. All patients were treated under general anesthesia and intravenous systemic heparinization. The preferred guiding catheter and microguidewire were the 6-F Envoy catheter (Cordis Neurovascular, Miami Lakes, FL) and the 0.014-inch Transcend microguidewire (Boston Scientific), respectively. The stent sizes used were 4 mm by 20 mm to 4.5 mm by 30 mm. Catheterization of the aneurysm was achieved following the deployment of the stent. Occasionally, the aneurysm was accessed with an SL 10 microcatheter (Boston Scientific) prior to the deployment of the stent. In these instances, the first framing coil was deployed, but not detached, before stent implantation.

Following stent deployment, coiling was performed in the usual fashion. In three patients, there was a four to six week interval between the stent implantation and coiling procedure to secure the stability of the neck-supporting struts. Both bare-platinum or surface-modified coils were used for embolization, including bare platinum coils (Micrus (Micrus Endovascular, San Jose, CA), GDC (Boston Scientific), and Trufill (Cordis Neurovascular)) for 16 patients and surface-modified coils (Matrix (Boston Scientific), Cerecyte (Micrus Endovascular)) for eight patients. Hydrocoils (Microvention/Terumo, Aliso Viejo, CA) were used in eight patients.

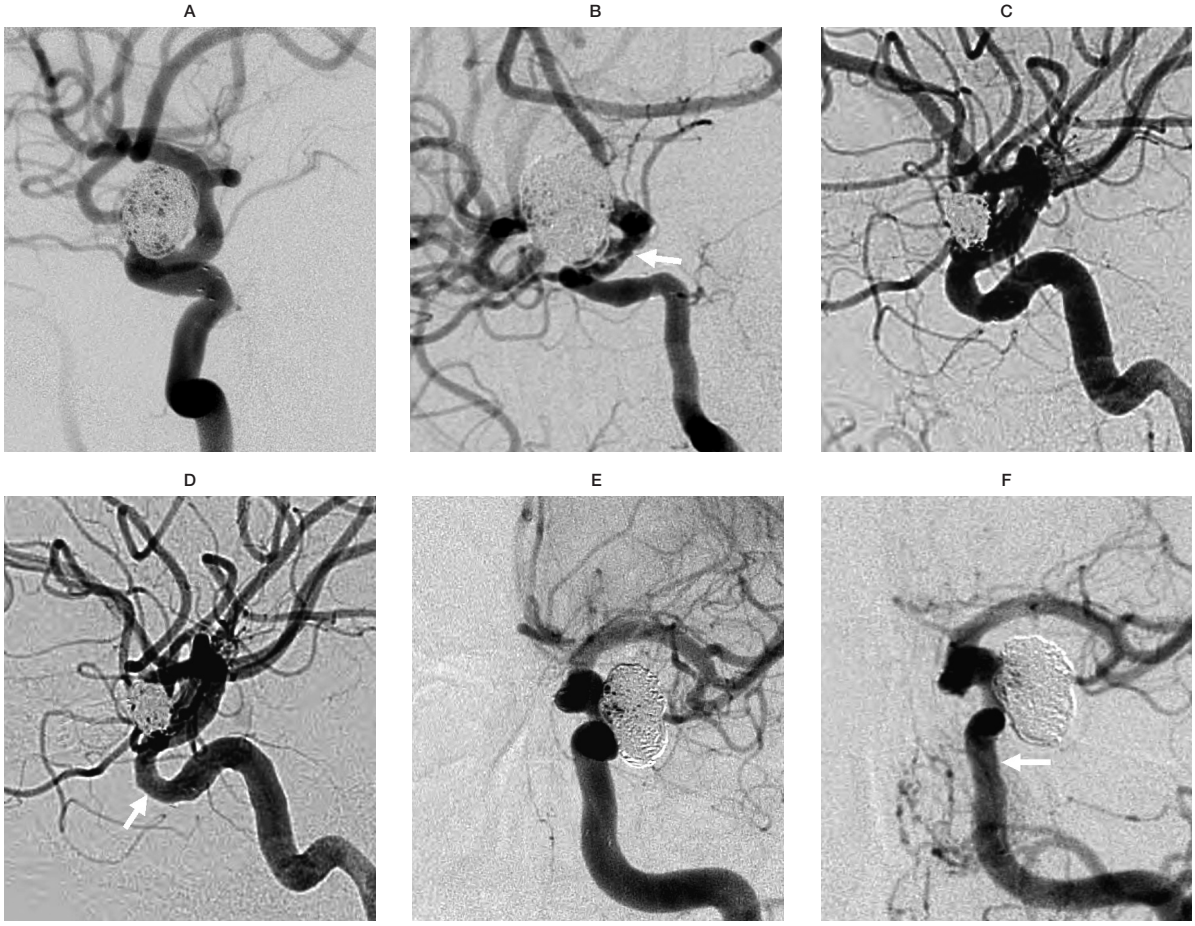
An angiogram was routinely obtained 30 minutes after completion of embolization procedure in order to rule out acute in-stent thrombosis. Intra-arterial local abciximab (ReoPro; Eli Lilly, Indianapolis, IN) was given at

Table 2 **Initial angiographic and follow-up results of embolization in patients with intimal thickening and without intimal thickening.**

Intimal thickening	Immediate result by Roy classification	Follow-up result		
		Improved	Unchanged	Worse
Yes	I (n=0)	0	0	0
	II (n=0)	0	0	0
	III (n=6)	6	0	0
No	I (n=7)	0	7	0
	II (n=6)	1	2	3
	III (n=13)	6	3	4

Table 3 **Univariate predictors of the occurrence of intimal thickening.**

Factors	Number of intimal thickening / Number in the subgroup	P value
Younger age (≤ 55)	6 / 11 (54.5%)	0.019
Presence of atherosclerotic risk factors	2 / 12 (16.7%)	0.631
Ruptured aneurysm	1 / 4 (25.0%)	1.000
Larger aneurysm (> 10 mm)	2 / 11 (18.2%)	1.000
Anterior circulation	4 / 21 (19.1%)	1.000
Stenting of small arteries	0 / 9 (0%)	0.150
Initial Roy class 3	6 / 19 (31.6%)	0.059
Longer stent (30 mm)	1 / 8 (12.5%)	1.000
Bioactive or Hydrocoils	4 / 16 (25%)	0.654
Angiographic improvement of aneurysm filling on follow-up	6 / 13 (46.2%)	0.002



the earliest sign of platelet aggregation on the stent. Otherwise, systemic heparinization was reversed using protamine sulfate before the patient was transferred to the neuro intensive care unit. Patients were placed on a maintenance dosage of dual antiplatelet therapy following the procedure and indefinitely thereafter. All patients were encouraged to undergo a six-month follow-up angiography.

Angiographic evaluation and analysis

Two experienced neuroradiologists (D.H.L, A.A) independently reviewed the angiographic images obtained immediately post-treatment and at six-month follow-up. Consensus was achieved if there was any discrepancy in the interpretation of the angiographic results.

The initial post-treatment results were classified based on the classification defined by Roy et Al[7]. Class 1 designated complete occlusion of the sac without residual filling, while classes 2 and 3 meant residual filling of the neck and the aneurysm sac, respectively.

The six-month follow-up angiographic findings were analyzed in terms of the stability of embolized aneurysms, recanalization, and any change in the lumen of the parent artery. The follow-up status of the aneurysm was categorized as 'improved' (decrease of residual neck and/or sac filling), 'unchanged' (no angiographic change from the initial status), or 'worse' (increase of aneurysm filling including recanalization). The Raymond and Roy classification was also applied in the analysis of the follow-up angiographic results.

'Intimal thickening' was determined based on the diameter changes in the parent artery between the immediate post-treatment angiogram and the follow-up angiogram. Any conceivable change in the caliber in consensus was appreciated as 'intimal thickening' regardless of the degree of the stenosis. In cases with the intimal thickening, the percentage change of lumen diameter was calculated using the WASID method[8]. A change in the parent artery diameter greater than 50% was defined as 'sig-

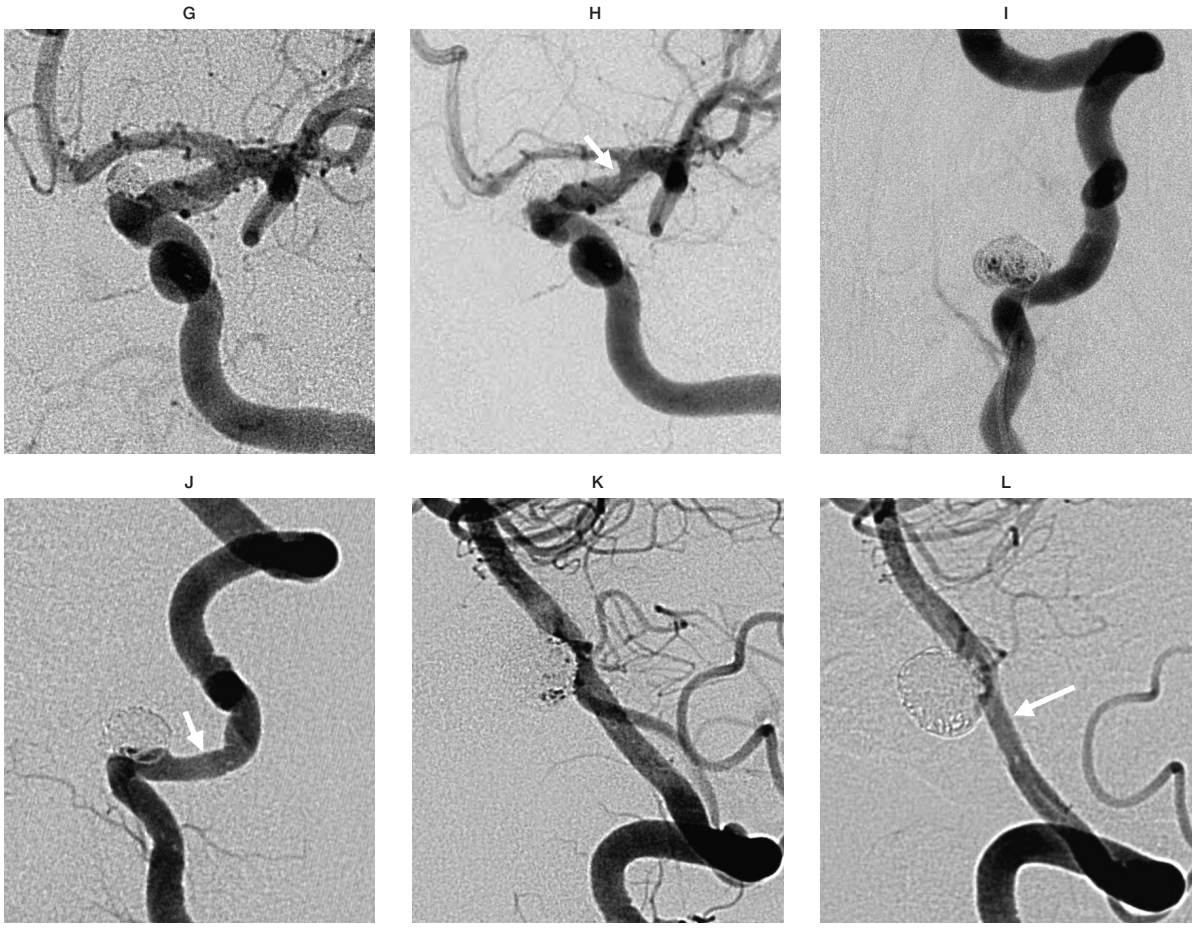


Figure 1 Representative angiographic images of the six cases show in-stent stenosis on follow-up. Pictures at the left side are angiograms obtained immediately after completion of the stent-assisted coil embolization; those at the left side are angiograms taken at the six-month follow-up. The white arrow in each follow-up image indicates the site of in-stent stenosis. Note the improvement of the follow-up angiographic embolization result in every case. A 43-year-old woman with an ophthalmic segment ICA aneurysm was treated with coil embolization after stenting, thus leaving some residual sac filling (A). A long-segment, irregular luminal stenosis is noted on her follow-up angiogram resulting in decreased antegrade flow. The residual sac filling has disappeared (B). A 47-year-old woman with another ophthalmic segment ICA aneurysm was treated with stent-assisted, coil embolization. Residual aneurysm sac filling is not well-demonstrated in this view (C). Follow-up angiogram shows diffuse but mild stenosis of the mid-portion of the stented segment (D). The immediate post-treatment angiogram of a clinoid segment ICA aneurysm in a 47-year-old woman shows residual filling of contrast media (E). The residual sac has almost gone, but on follow-up there is a small amount of residual filling near the neck portion. Diffuse but mild stenosis is noted, especially in the lateral aspect of the proximal stented segment (F). A 26-year-old woman with a small ophthalmic artery origin aneurysm, has a small residual sac filling after embolization (G). Short-segment, concentric stenosis is noted at the distal end of the stent on her follow-up angiogram, while the residual sac filling is not (H). A 55-year-old woman with a vertebral artery aneurysm, experienced significant residual filling of the sac after coiling (I). The residual sac filling has gone on follow-up angiogram and the distal segment of the stented artery shows eccentric but diffuse stenosis (J). A 52- year-old woman with an aneurysm arising from the fenestrated basilar trunk, was treated with stent-assisted coiling, thus leaving a residual sac in the lower portion of the aneurysm (K). Disappearance of the residual sac and diffuse stenosis of the proximal segment of the parent artery are noted on her follow-up angiogram (L).

nificant intimal thickening' as it is identical to the in-stent stenosis, a definition of which has been used by previous authors [6,9].

SPSS for Windows (version 12.0; SPSS Inc., Chicago, IL) was used for the statistical analyses. Fisher exact tests were used to test the relationship between the occurrence of the intimal thickening and the following variables: clinical presentation (ruptured or unruptured); patient age (≤ 55 and >55); presence of any risk factor for atherosclerosis (yes and no); aneurysm location (anterior or posterior circulation); aneurysm size (maximum diameter of ≤ 10 mm and >10 mm); length of the stent used

(20 mm and 30 mm); coil types used (bare platinum coils, bioactive coils, and Hydrocoils); stent placement in small cortical arteries such as the proximal anterior cerebral artery (yes and no); Roy classification of the angiographic result of embolization (classes 1, 2, and 3); and the follow-up angiographic results of the embolized aneurysm (improved, unchanged, or worse). Separate logistic regression analysis was performed for independent variables showing a *p* value of less than 0.15 for the multivariate analysis. *P* values less than 0.05 were considered to be significant.

Results

The Raymond and Roy classification of the initial angiographic embolization result was class 1 (complete occlusion) in seven study patients (21.9%), class 2 (residual neck) in six patients (18.8%), and class 3 (residual sac) in 19 patients (59.4%). Follow-up angiogram was obtained after an average of 6.1 months (range, 3-15 months). There were six cases (18.8%) of 'intimal thickening' at the stented segment of the parent artery (figure 1). The average stenosis was 25.8% (range, 7-52%). Among these patients, 'significant intimal thickening' was noted in one patient (3.1%) (figure 1b). The degree of angiographic occlusion on follow-up studies was 'improved' in 13 patients (40.6%), 'unchanged' in 12 patients (37.5%), and 'worse' in seven (21.9%). The follow-up results of both the intimal-thickening group and non-intimal-thickening group are tabulated in Table 2. The Raymond and Roy classification of the six-month follow-up angiography results was class 1 in 15 patients (46.9%), class 2 in six (18.8%), and class 3 in 11 patients (34.4%).

The influence of univariate indicators on the incidence of the intimal thickening is listed in detail in Table 3. Statistically significant results were noted with the variables of patient age (≤ 55) and follow-up angiographic results ('improved'). All cases (100%) that showed intimal thickening were associated with the improvement of angiography results on the six-month follow-up, while only seven (26.9%) of the cases without intimal thickening had an improvement in the degree of aneurysm obliteration on follow-up ($p < 0.005$).

However, multivariate analysis, including patient age, stenting of small cortical arteries, initial Raymond and Roy classification, and angiographic improvement of aneurysm filling on follow-up, failed to show any significant predictor of the intimal thickening.

Discussion

Although 'significant intimal thickening' was infrequent, there was a relatively high incidence of 'intimal thickening' in the parent artery implanted with a Neuroform stent when we consider any kind of luminal narrowing, which is conceivable on the angiogram. The presence of the intimal thickening was associated favorably with an improvement of the degree of aneurysm obliteration on six-month follow-up angiographic studies.

The occurrence of in-stent restenosis (ISR) after coronary artery stenting is relatively well understood [10,11]. The incidence of ISR in the coronary artery has been reported to be as high as 10-60%, especially in patients with high risk factors, such as diabetes, long-segment lesion, or small caliber artery, etc.[12,13]. Angiographic patterns of ISR can be classified as focal, diffuse, diffuse proliferative and total occlusion of the parent artery [9]. It is generally thought that the ISR following stenting of a stenotic coronary artery is due primarily to neointimal hyperplasia as a local response to the arterial wall (mostly at the endothelial layer) injury and which can be combined with progression of the primary atherosclerotic disease. Histologically, neointimal hyperplasia is a chronic change in the arterial wall manifested as diffuse thickening of the fibro-cellular layer located between the endothelial lining and the internal elastic lamina [12,13].

ISR is a multifactorial complex process that can be divided into two phases, i.e. early and late, of reaction. The early phase spans from a period of days to weeks and features reorganization of thrombi and some inflammatory reaction provoked by the aggregated platelets, fibrin, and leukocytes. The late phase spans a period of from weeks to months. The major actors in this late phase, the smooth muscle cells migrate to the intima from the media and then undergo proliferation which results in thickening of the intima. The cellularity of the thickened intima becomes low with increased content of the extracellular matrix to become more stable [11,13]. However, the same mechanism cannot be inferred from ISR of coronary stents to the intimal thickening provoked by Neuroform

stents. Intuitively, the intimal injury which occurs during the implantation of the self expandable stent for the stent-assisted aneurysm embolization seems to be less than the injury that occurs during the high-pressure inflation of the balloon-expandable stents. Furthermore, in our cases, there was no angiographic evidence of atherosclerosis in Neuroform-stented parent artery, and only one-third of our study patients had atherosclerotic risk factors. In a post-mortem histological case report, Lopez et Al showed a certain degree of intimal thickening on the follow-up angiogram obtained three months after Neuroform stent placement [14]. The authors observed that the main component of the thickened intimal layer was caused by intimal hyperplasia while the endothelium-covered stent struts abutted the internal elastic lamina. Unfortunately, it is unclear from the report whether or not smooth muscle cells contributed to the intimal thickening.

Then what is the possible mechanism of the intimal thickening following the implantation of Neuroform stents? Although the injury initiated by the self-expanding process of the stent is usually mild due to its relatively weak radial force [5], it may, nonetheless, cause an injury to the intima as well as denudation of the friable endothelial layer, both of which can be exaggerated if the deployment procedure was traumatic. Then the exposed subendothelial structures can be good foci for the adhesion and aggregation of platelets similar to the reaction that can be observed during the coronary stenting procedure. Although there is only a small chance of this occurring when compared with that which can be seen after the balloon-expandable coronary stenting procedure, the implantation of a Neuroform stent can also deform the geometry of the vessel to some degree, thereby resulting in a change of vascular wall shear stress. Vascular wall shear stress is a major factor in the maintenance of endothelial integrity and the prevention of thrombosis [15]. In addition to the parent artery deformation, the indentation of the arterial lumen by the stent struts might have another role in the disturbance of even laminar flow at the stented segment, which could affect change in the local wall shear stress near the struts.

This thrombosis and intimal injury may progress to intimal hyperplasia, the degree of which can vary among different patients. In our study, the individual arterial tissue response to the stent varied, despite the absence of significant atherosclerotic risk factors and the uniformity of antiplatelet treatment. It seems likely that, in some patients, the arterial response to the relatively atraumatic deployment step of the Neuroform stent insertion, can be enough to cause some degree of intimal thickening and occasionally significant intimal thickening.

We postulate that if the tissue response to the implantation of a Neuroform stent is aggressive enough to cause conceivable intimal thickening, the same tissue response to the coil mass in the aneurysm sac might have a beneficial late effect in improving the degree of the obliteration of the aneurysm over time. Although not mentioned in their reports on in-stent stenosis of Neuroform stents [6,16], some of their representative figures clearly showed the disappearance of the residual aneurysm sac on the follow-up angiograms. Interestingly, the same finding was observed in Lopez et Al's case report [14] in which the aneurysm sac showed a decrease in size on follow-up angiograms. They observed an accumulation of fibro-elastic tissue around the neck on microscopy even though there was no coil in the aneurysm.

Debrun described this competing behavior of the body's response to the implanted stents and coils [17]. Intimal hyperplasia resulting from local thrombosis and denudation of the intimal layer is necessary for the optimal healing of an aneurysm. However, this same tissue response is also the major cause of intimal thickening in the parent artery. We believe that what we observed in our series is a synchronous relationship between the excessive endothelial response to the Neuroform stent which cause the intimal thickening and the improvement in aneurysm obliteration on follow-up angiogram by virtue of the excessive tissue response to the coil mass. The mechanism of the intimal thickening and its variability between patients might be also responsible for improving the healing in embolized aneurysms. If a patient is prone to excessive tissue response after coiling, the chance of the aneurysm healing might be high.

Another factor that needs to be considered is the possible effect of Neuroform stent on the intra-aneurysmal flow. Stents may change the biomechanical characteristics of both an aneurysm and the parent artery. It is said that stents can decrease the aneurysmal flow which may lead to a decrease in the aneurysmal wall shear stress and consequent thrombosis [18]. This

mechanism could facilitate the healing process of the embolized aneurysm with time. However, it cannot fully explain the occurrence of the phenomenon in our particular patients.

We were unable to find any significant implication of the more frequent occurrence of intimal thickening in younger patients. Does this indicate more frequent and exuberant tissue response to a stent and/or coils in younger patients? Conversely, the presence of atherosclerotic risk factors did not affect the occurrence of intimal thickening. Even the size discrepancy between the stent diameter used and the diameter of the artery, and use of bioactive coils or Hydrocoils, did not seem to have any influence on its occurrence.

Our series includes several large and/or partially thrombosed cavernous or paraclinoid aneurysms, which might explain the relatively high number of Roy class 3. This might have been a potential source of bias. Another limitation of our study is the small number of patients which we thought might be a possible reason for the negative results of our multivariate analysis.

Conclusions

Deployment of a Neuroform stent can produce a variable degree of intimal thickening at the stented parent artery without any underlying atherosclerotic risk factors. In our limited series, the occurrence of the intimal thickening was more prevalent in younger patients and was often associated with an improvement in aneurysm obliteration rates on follow-up angiogram.

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